INVENTOR SEARCH

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=> fil medl pascal caba wpix biotechno biosis lifesci confsci capl dissabs embase;
d que l12
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FILE 'PASCAL' ENTERED AT 11:46:58 ON 27 DEC 2006
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=> d que l6; d que l15
L1          619 SEA GILULA N/AU OR GILULA NB/AU OR GILULA N B/AU OR GILULA
                NORTON?/AU
L2          673 SEA CRAVATT B/AU OR CRAVATT BF/AU OR CRAVATT B F/AU OR CRAVATT
                BENJAMIN?/AU
L3         2601 SEA LERNER R/AU OR LERNER RA/AU OR LERNER R A/AU OR LERNER
                RICH?/AU
L6           35 SEA L1 AND L2 AND L3



L1          619 SEA GILULA N/AU OR GILULA NB/AU OR GILULA N B/AU OR GILULA
                NORTON?/AU
L2          673 SEA CRAVATT B/AU OR CRAVATT BF/AU OR CRAVATT B F/AU OR CRAVATT
                BENJAMIN?/AU
L3         2601 SEA LERNER R/AU OR LERNER RA/AU OR LERNER R A/AU OR LERNER
                RICH?/AU
L5         1164 SEA FAAH
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L10         1837 SEA FATTY-ACID AMIDE HYDROLASE
L15           25 SEA ((L1 AND (L2 OR L3)) OR (L2 AND L3)) AND (L5 OR L10)


=> s l6,l15
L28           41 (L6 OR L15)

=> s l28 not l22,l25
L29           40 L28 NOT (L22 OR L25)

=> dup rem l29
PROCESSING COMPLETED FOR L29
L30           14 DUP REM L29 (26 DUPLICATES REMOVED)
                 ANSWERS '1-5' FROM FILE MEDLINE
                 ANSWERS '6-7' FROM FILE WPIX
                 ANSWERS '8-11' FROM FILE BIOSIS
                 ANSWER '12' FROM FILE CONFSCI
                 ANSWERS '13-14' FROM FILE CAPLUS

=> d iall 1-5; d iall abeq tech 6-7; d iall 8-12; d ibib ed abs hitind 13-14; fil
hom

L30  ANSWER 1 OF 14      MEDLINE on STN                    DUPLICATE 2
ACCESSION NUMBER:    1998226723      MEDLINE  Full-text
DOCUMENT NUMBER:     PubMed ID: 9560184
TITLE:               Chemical requirements for inhibition of gap junction
                     communication by the biologically active lipid oleamide.
AUTHOR:              Boger D L; Patterson J E; Guan X; Cravatt B F;
                     Lerner R A; Gilula N B
CORPORATE SOURCE:    Department of Chemistry, The Scripps Research Institute,
                     10550 North Torrey Pines Road, La Jolla, CA 92037, USA..
                     boger@scripps.edu
CONTRACT NUMBER:     CA42056 (NCI)
SOURCE:              Proceedings of the National Academy of Sciences of the
                     United States of America, (1998 Apr 28) Vol. 95, No. 9, pp.
                     4810-5.
                     Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199806
ENTRY DATE:          Entered STN: 11 Jun 1998
                     Last Updated on STN: 11 Jun 1998
                     Entered Medline: 4 Jun 1998
ABSTRACT:
Oleamide is an endogenous fatty acid primary amide that possesses
sleep-inducing properties in animals and has been shown to effect serotonergic
systems and block gap junction communication in a structurally specific manner.
Herein, the structural features of oleamide required for inhibition of the gap
junction-mediated chemical and electrical transmission in rat glial cells are
defined.  The effective inhibitors fall into two classes of fatty acid primary
amides of which oleamide and arachidonamide are the prototypical members.  Of
these two, oleamide constitutes the most effective, and its structural
requirements for inhibition of the gap junction are well defined.  It requires
a chain length of 16-24 carbons of which 16-18 carbons appears optimal, a
polarized terminal carbonyl group capable of accepting but not necessarily
donating a hydrogen bond, a Delta9 cis double bond, and a hydrophobic methyl
terminus.  Within these constraints, a range of modifications are possible,
many of which may be expected to improve in vivo properties.  A select set of
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agents has been identified that serves both as oleamide agonists and as inhibitors of **fatty acid amide hydrolase**
, which is responsible for the rapid inactivation of oleamide.
CONTROLLED TERM: Amidohydrolases: ME, metabolism
Animals
*Cell Communication: DE, drug effects
Cells, Cultured
*Gap Junctions: DE, drug effects
Isomerism
Neuroglia: DE, drug effects
Oleic Acids: CH, chemistry
Oleic Acids: ME, metabolism
*Oleic Acids: PD, pharmacology
Rats
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Structure-Activity Relationship
CAS REGISTRY NO.: 301-02-0 (oleylamide)
CHEMICAL NAME: 0 (Oleic Acids); EC 3.5. (Amidohydrolases); EC 3.5.1.- (**fatty-acid amide hydrolase**)

L30 ANSWER 2 OF 14 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1998075090 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 9412472
TITLE: The sleep-inducing lipid oleamide deconvolutes gap junction communication and calcium wave transmission in glial cells.
AUTHOR: Guan X; **Cravatt B F**; Ehring G R; Hall J E; Boger D L; **Lerner R A**; **Gilula N B**
CORPORATE SOURCE: Department of Cell Biology, The Scripps Research Institute, La Jolla, California 92037, USA.
SOURCE: The Journal of cell biology, (1997 Dec 29) Vol. 139, No. 7, pp. 1785-92.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 17 Feb 1998
Last Updated on STN: 17 Feb 1998
Entered Medline: 5 Feb 1998

ABSTRACT:
Oleamide is a sleep-inducing lipid originally isolated from the cerebrospinal fluid of sleep-deprived cats. Oleamide was found to potently and selectively inactivate gap junction-mediated communication between rat glial cells. In contrast, oleamide had no effect on mechanically stimulated calcium wave transmission in this same cell type. Other chemical compounds traditionally used as inhibitors of gap junctional communication, like heptanol and 18beta-glycyrrhetinic acid, blocked not only gap junctional communication but also intercellular calcium signaling. Given the central role for intercellular small molecule and electrical signaling in central nervous system function, oleamide- induced inactivation of glial cell gap junction channels may serve to regulate communication between brain cells, and in doing so, may influence higher order neuronal events like sleep induction.
CONTROLLED TERM: Animals
*Calcium: ME, metabolism
*Cell Communication: DE, drug effects
Cell Line

Connexin 43: ME, metabolism
Connexins: GE, genetics
Connexins: ME, metabolism
Cricetinae
Fluorescent Dyes: ME, metabolism
Gap Junctions: DE, drug effects
*Gap Junctions: PH, physiology
Isoquinolines: ME, metabolism
Neuroglia: CY, cytology
Neuroglia: DE, drug effects
*Neuroglia: ME, metabolism
*Oleic Acids: PD, pharmacology
Phosphorylation
Rats
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Sleep
Structure-Activity Relationship

CAS REGISTRY NO.: 301-02-0 (oleylamide); 7440-70-2 (Calcium); 77944-88-8 (lucifer yellow)

CHEMICAL NAME: 0 (Connexin 43); 0 (Connexins); 0 (Fluorescent Dyes); 0 (Isoquinolines); 0 (Oleic Acids); 0 (connexin 32)

L30 ANSWER 3 OF 14 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 1998112399 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 9452020

TITLE: **Fatty acid amide hydrolase**, the degradative enzyme for anandamide and oleamide, has selective distribution in neurons within the rat central nervous system.

AUTHOR: Thomas E A; **Cravatt B F**; Danielson P E; **Gilula N B**; Sutcliffe J G

CORPORATE SOURCE: Department of Molecular Biology, The Scripps Research Institute, La Jolla, California 92037, USA.

CONTRACT NUMBER: GM32355 (NIGMS)

SOURCE: Journal of neuroscience research, (1997 Dec 15) Vol. 50, No. 6, pp. 1047-52.
Journal code: 7600111. ISSN: 0360-4012.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199803

ENTRY DATE: Entered STN: 12 Mar 1998
Last Updated on STN: 12 Mar 1998
Entered Medline: 4 Mar 1998

ABSTRACT:

Fatty acid amide hydrolase (***FAAH***) is a membrane-bound enzyme activity that degrades neuromodulatory fatty acid amides, including oleamide and anandamide. A single 2.5-kb ***FAAH*** mRNA is distributed throughout the rat CNS and accumulates progressively between embryonic day 14 and postnatal day 10, remains high until postnatal day 30, then decreases into adulthood. **FAAH** enzymatic activity, as measured in dissected brain regions, was well correlated with the distribution of its messenger RNA. In situ hybridization revealed profound distribution of **FAAH** mRNA in neuronal cells throughout the CNS. The most prominent signals were detected in the neocortex, hippocampal formation, amygdala, and cerebellum. The **FAAH** distribution in the CNS suggests that degradation of neuromodulatory fatty acid amides at their sites of action

influences their effects on sleep, euphoria, and analgesia.
CONTROLLED TERM: Check Tags: Male
*Amidohydrolases: AN, analysis
Amidohydrolases: GE, genetics
Animals
*Arachidonic Acids: ME, metabolism
Blotting, Northern
*Central Nervous System: CH, chemistry
In Situ Hybridization
*Neurons: ME, metabolism
*Oleic Acids: ME, metabolism
*RNA, Messenger: AN, analysis
Rats
Rats, Sprague-Dawley
Research Support, U.S. Gov't, P.H.S.
CAS REGISTRY NO.: 301-02-0 (oleylamide); 94421-68-8 (anandamide)
CHEMICAL NAME: 0 (Arachidonic Acids); 0 (Oleic Acids); 0 (RNA, Messenger); EC 3.5. (Amidohydrolases); EC 3.5.1.- (**fatty-acid amide hydrolase**)

L30 ANSWER 4 OF 14 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 97055939 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 8900284
TITLE: Molecular characterization of an enzyme that degrades neuromodulatory fatty-acid amides.
AUTHOR: **Cravatt B F**; Giang D K; Mayfield S P; Boger D L; **Lerner R A**; **Gilula N B**
CORPORATE SOURCE: Department of Chemistry, The Scripps Research Institute, La Jolla, California 92307, USA.
SOURCE: Nature, (1996 Nov 7) Vol. 384, No. 6604, pp. 83-7.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U72497
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 10 Dec 1996

ABSTRACT:
Endogenous neuromodulatory molecules are commonly coupled to specific metabolic enzymes to ensure rapid signal inactivation. Thus, acetylcholine is hydrolysed by acetylcholine esterase and tryptamine neurotransmitters like serotonin are degraded by monoamine oxidases. Previously, we reported the structure and sleep-inducing properties of cis-9-octadecenamide, a lipid isolated from the cerebrospinal fluid of sleep-deprived cats. cis-9-Octadecenamide, or oleamide, has since been shown to affect serotonergic systems and block gap-junction communication in glial cells (our unpublished results). We also identified a membrane-bound enzyme activity that hydrolyses oleamide to its inactive acid, oleic acid. We now report the mechanism-based isolation, cloning and expression of this enzyme activity, originally named oleamide hydrolase, from rat liver plasma membranes. We also show that oleamide hydrolase converts anandamide, a fatty-acid amide identified as the endogenous ligand for the cannabinoid receptor, to arachidonic acid, indicating that oleamide hydrolase may serve as the general inactivating enzyme for a growing family of bioactive signalling molecules, the fatty-acid amides. Therefore we will hereafter refer to oleamide hydrolase as **fatty-acid amide** ***hydrolase*** , in recognition of the plurality of fatty-acid amides that the enzyme can accept as substrates.

CONTROLLED TERM: *Amides: ME, metabolism
Amidohydrolases: GE, genetics
Amidohydrolases: IP, isolation & purification
*Amidohydrolases: ME, metabolism
Amino Acid Sequence
Animals
Arachidonic Acids: ME, metabolism
Blotting, Northern
Blotting, Southern
COS Cells
Cell Membrane: EN, enzymology
Chromatography, Affinity
Cloning, Molecular
*Fatty Acids: ME, metabolism
*Liver: EN, enzymology
Molecular Sequence Data
Rats
Sequence Homology, Amino Acid
CAS REGISTRY NO.: 94421-68-8 (anandamide)
CHEMICAL NAME: 0 (Amides); 0 (Arachidonic Acids); 0 (Fatty Acids); EC 3.5. (Amidohydrolases); EC 3.5.1.- (**fatty-acid amide hydrolase**)

L30 ANSWER 5 OF 14 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 95288645 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 7770779
TITLE: Chemical characterization of a family of brain lipids that induce sleep.
AUTHOR: **Cravatt B F**; Prospero-Garcia O; Siuzdak G; **Gilula N B**; Henriksen S J; Boger D L; **Lerner R A**
CORPORATE SOURCE: Department of Chemistry, Scripps Research Institute, La Jolla, CA 92307, USA.
CONTRACT NUMBER: 1 S10 RR07273-01 (NCRR)
SOURCE: Science, (1995 Jun 9) Vol. 268, No. 5216, pp. 1506-9. Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199507
ENTRY DATE: Entered STN: 13 Jul 1995
Last Updated on STN: 6 Feb 1998
Entered Medline: 5 Jul 1995

ABSTRACT:
A molecule isolated from the cerebrospinal fluid of sleep-deprived cats has been chemically characterized and identified as cis-9,10-octadecenoamide. Other fatty acid primary amides in addition to cis-9,10-octadecenoamide were identified as natural constituents of the cerebrospinal fluid of cat, rat, and human, indicating that these compounds compose a distinct family of brain lipids. Synthetic cis-9,10-octadecenoamide induced physiological sleep when injected into rats. Together, these results suggest that fatty acid primary amides may represent a previously unrecognized class of biological signaling molecules.

CONTROLLED TERM: Animals
*Brain Chemistry
Cats
*Cerebrosides: CF, cerebrospinal fluid
Cerebrosides: CH, chemistry
Cerebrosides: PD, pharmacology

Humans
*Lipids: CF, cerebrospinal fluid
Lipids: CH, chemistry
Lipids: PD, pharmacology
Magnetic Resonance Spectroscopy
Molecular Weight
*Oleic Acids: CF, cerebrospinal fluid
Oleic Acids: CH, chemistry
Oleic Acids: PD, pharmacology
Rats
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Signal Transduction
*Sleep
Sleep: DE, drug effects
Spectrometry, Mass, Fast Atom Bombardment
Spectrophotometry, Infrared
Spectrum Analysis, Mass
CAS REGISTRY NO.: 301-02-0 (oleylamide)
CHEMICAL NAME: 0 (Cerebrosides); 0 (Lipids); 0 (Oleic Acids)

L30 ANSWER 6 OF 14 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
DUPLICATE 1
ACCESSION NUMBER: 1999-347603 [29] WPIX
DOC. NO. CPI: C1999-102314 [29]
TITLE: Oleamide agonists and **fatty acid amide hydrolase** inhibitors
DERWENT CLASS: B05
INVENTOR: BOGER D; BOGER D L; **CRAVATT B; CRAVATT B F; GILULA N; GILULA N B; LERNER R; LERNER R A**; BOGER L; CRAVATT F; GILULA B; LERNER A
PATENT ASSIGNEE: (SCRI-C) SCRIPPS RES INST
COUNTRY COUNT: 81

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 9926584	A2	19990603	(199929)*	EN	106[22]	A61K000-00
AU 9915973	A	19990615	(199944)	EN		A61K000-00
EP 1039902	A2	20001004	(200050)	EN		A61K031-40
US 6251931	B1	20010626	(200138)	EN		A61K031-40
AU 740588	B	20011108	(200176)	EN		A61K031-40
JP 2001523695	W	20011127	(200204)	JA	105	C07C047-21
EP 1039902	B1	20060308	(200618)	EN		
DE 69833791	E	20060504	(200634)	DE		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9926584 A2		WO 1998-US24913	19981124
EP 1039902 A2		EP 1998-960363	19981124
EP 1039902 B1		EP 1998-960363	19981124
EP 1039902 A2		WO 1998-US24913	19981124

US 6251931 B1	WO 1998-US24913 19981124
JP 2001523695 W	WO 1998-US24913 19981124
EP 1039902 B1	WO 1998-US24913 19981124
AU 9915973 A	AU 1999-15973 19981124
AU 740588 B	AU 1999-15973 19981124
JP 2001523695 W	JP 2000-521789 19981124
US 6251931 B1	US 2000-529909 20000419
DE 69833791 E	DE 1998-633791 19981124
DE 69833791 E	EP 1998-960363 19981124
DE 69833791 E	WO 1998-US24913 19981124

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
AU 740588	B	Previous Publ	AU 9915973	A
AU 9915973	A	Based on	WO 9926584	A
EP 1039902	A2	Based on	WO 9926584	A
US 6251931	B1	Based on	WO 9926584	A
AU 740588	B	Based on	WO 9926584	A
JP 2001523695	W	Based on	WO 9926584	A
EP 1039902	B1	Based on	WO 9926584	A
DE 69833791	E	Based on	EP 1039902	A
DE 69833791	E	Based on	WO 9926584	A

PRIORITY APPLN. INFO: US 1997-977493 19971124
US 2000-529909 20000419

INT. PATENT CLASSIF.:

MAIN: A61K; A61K031-40; C07C047-21

SECONDARY: A01N043-36; A61K031-16; A61K031-201; A61K031-231; A61K031-27; C07C233-00; C07C233-09; C07C233-10; C07C233-11; C07C233-20; C07C233-49; C07C235-06; C07C235-28; C07C235-76; C07C237-22; C07C243-30; C07C245-14; C07C259-06; C07C271-10; C07C275-20; C07C323-60; C07C327-32; C07C049-227; C07C057-12; C07C059-00; C07C069-58; C07D207-00; C07D295-00; C07D295-18

IPC ORIGINAL: A61K0031-40 [I,A]; A61P0029-00 [I,A]; A61K0031-40 [I,A]; A61P0029-00 [I,A]

BASIC ABSTRACT:

WO 1999026584 A2 UPAB: 20060315

NOVELTY - Compounds with oleamide agonist activity are used for inhibiting gap junction-mediated chemical and electrical transmission in glial cells.

DETAILED DESCRIPTION - The compounds are oleamide analogs of formula (I): X = cis- or trans-1,2-ethenylene, ethynylene, trans-1,2- cyclopropylene or trans-2,3-(1-oxa)cyclopropylene; Y = CH2, CH(CH3), C(CH3)2, O, NH, CH(SH), CHSAc, CH(OH), CHCl, C(O), C(O)CH2, CH2NHC(O) or CH2N(CH3)C(O); R1 = H, NH2, OH, MeNH, Me2N, EtNH, Et2N, CH=CHCH2NH, n-propyl-NH, i-propyl-NH, cyclopropyl-NH, i-propyl-NMe, butyl-NH, pyrrolidine, phenyl-NH, phenyl(CH2)3NH, HONH, MeONMe, NH2NH, CH3O, CH3CH2O, CH3(CH2)O, Me2CHCH2O, H, CF3, BrCH2, ClCH2, N2CH, HOCH2CH2NH, (HOCH2CH2)2N, HOCH2CH2CH2NH or HOCH2CH(OAc)CH2O; R2 = CH3, (CH2)2CH3, (CH2)4CH3, (CH2)6CH3, CH2OCH3, CH2OH, CONH2 or CO2H; n, m = 0-15, and n+m = 11-15; provided that:

(i) if Y = CH3, n = 4, m = 7 and R2 = CH3, then R1 is not CF3 or H;

(ii) if Y = CH2, n = 5, m = 7 and R2 = CH3, then R1 is not CF3, CH2Cl, NHOH, C(O)NH2, CN2 or C(O)OEt; (iii) if Y = CH2Cl, n = 4, m = 7 and R2 = CH3, then R1 is not NH2; (iv) if Y = CH(OH), n = 4, m = 7 and R2 = CH3, then R1 is not NH2; (v) if Y = C(O), n = 4, m = 7 and R2 = CH3 , then R1 is not NH2 or CH3CH2O; and

(vi) if Y = CH2, n = 4-9, m = 4-7 and R2 = CH3, then R1 is not NH2 or OH.

ACTIVITY - Neuromodulatory; sleep disorders; mood disorders; analgesia.
MECHANISM OF ACTION - Oleamide agonist; **fatty acid amide hydrolase** inhibitor.
USE - Analogs for oleamide, an endogenous fatty acid primary amide that possesses sleep-inducing properties in animals known to affect serotonergic systems and blocking gap junction communication in a structurally specific manner. Used to inhibit gap junction-mediated chemical and electrical transmission in glial cells (claimed). Used to help distinguish among mechanisms for gap junction inhibition by oleamide and as new neuromodulatory agents applicable to sleep or mood disorders, analgesia, and disorders associated with higher neuronal function. Act as fluidity transmitters.
ADVANTAGE - Storage and release may be controlled in a manner analogous to that of short peptide hormones and messengers terminating in a primary amide.
MANUAL CODE: CPI: B07-A03; B10-B04B; B10-C02; B10-C04; B10-D01; B10-D03; B10-E04D; B10-F02; B10-G02; B14-C01; B14-J01; B14-J01B2

Member(0003)
ABEQ EP 1039902 A2 UPAB 20060315
NOVELTY - Compounds with oleamide agonist activity are used for inhibiting gap junction-mediated chemical and electrical transmission in glial cells.
DETAILED DESCRIPTION - The compounds are oleamide analogs of formula (I):
X = cis- or trans-1,2-ethenylene, ethynylene, trans-1,2-cyclopropylene or trans-2,3-(1-oxa)cyclopropylene;
Y = CH2, CH(CH3), C(CH3)2, O, NH, CH(SH), CHSAc, CH(OH), CHCl, C(O), C(O)CH2, CH2NHC(O) or CH2N(CH3)C(O);
R1 = H, NH2, OH, MeNH, Me2N, EtNH, Et2N, CH=CHCH2NH, n-propyl-NH, i-propyl-NH, cyclopropyl-NH, i-propyl-NMe, butyl-NH, pyrrolidine, phenyl-NH, phenyl(CH2)3NH, HONH, MeONMe, NH2NH, CH3O, CH3CH2O, CH3(CH2)O, Me2CHCH2O, H, CF3, BrCH2, ClCH2, N2CH, HOCH2CH2NH, (HOCH2CH2)2N, HOCH2CH2CH2NH or HOCH2CH(OAc)CH2O;
R2 = CH3, (CH2)2CH3, (CH2)4CH3, (CH2)6CH3, CH2OCH3, CH2OH, CONH2 or CO2H;
n, m = 0-15, and n+m = 11-15;
provided that:
(i) if Y = CH3, n = 4, m = 7 and R2 = CH3, then R1 is not CF3 or H;
(ii) if Y = CH2, n = 5, m = 7 and R2 = CH3, then R1 is not CF3, CH2Cl, NHOH, C(O)NH2, CN2 or C(O)OEt;
(iii) if Y = CH2Cl, n = 4, m = 7 and R2 = CH3, then R1 is not NH2;
(iv) if Y = CH(OH), n = 4, m = 7 and R2 = CH3, then R1 is not NH2;
(v) if Y = C(O), n = 4, m = 7 and R2 = CH3 , then R1 is not NH2 or CH3CH2O; and
(vi) if Y = CH2, n = 4-9, m = 4-7 and R2 = CH3, then R1 is not NH2 or OH.
ACTIVITY - Neuromodulatory; sleep disorders; mood disorders; analgesia.
MECHANISM OF ACTION - Oleamide agonist; **fatty acid amide hydrolase** inhibitor.
USE - Analogs for oleamide, an endogenous fatty acid primary amide that possesses sleep-inducing properties in animals known to affect serotonergic systems and blocking gap junction communication in a structurally specific manner. Used to inhibit gap junction-mediated chemical and electrical transmission in glial cells (claimed). Used to help distinguish among mechanisms for gap junction inhibition by oleamide and as new neuromodulatory agents applicable to sleep or mood disorders, analgesia, and disorders associated with higher neuronal function. Act as fluidity transmitters.
ADVANTAGE - Storage and release may be controlled in a manner analogous to that of short peptide hormones and messengers terminating in

a primary amide.

Member(0004)
ABEQ US 6251931 B1 UPAB 20060315
NOVELTY - Compounds with oleamide agonist activity are used for inhibiting gap junction-mediated chemical and electrical transmission in glial cells.
DETAILED DESCRIPTION - The compounds are oleamide analogs of formula (I):
X = cis- or trans-1,2-ethenylene, ethynylene, trans-1,2-cyclopropylene or trans-2,3-(1-oxa)cyclopropylene;
Y = CH2, CH(CH3), C(CH3)2, O, NH, CH(SH), CHSAc, CH(OH), CHCl, C(O), C(O)CH2, CH2NHC(O) or CH2N(CH3)C(O);
R1 = H, NH2, OH, MeNH, Me2N, EtNH, Et2N, CH=CHCH2NH, n-propyl-NH, i-propyl-NH, cyclopropyl-NH, i-propyl-NMe, butyl-NH, pyrrolidine, phenyl-NH, phenyl(CH2)3NH, HONH, MeONMe, NH2NH, CH3O, CH3CH2O, CH3(CH2)O, Me2CHCH2O, H, CF3, BrCH2, ClCH2, N2CH, HOCH2CH2NH, (HOCH2CH2)2N, HOCH2CH2CH2NH or HOCH2CH(OAc)CH2O;
R2 = CH3, (CH2)2CH3, (CH2)4CH3, (CH2)6CH3, CH2OCH3, CH2OH, CONH2 or CO2H;
n, m = 0-15, and n+m = 11-15;
provided that:
(i) if Y = CH3, n = 4, m = 7 and R2 = CH3, then R1 is not CF3 or H;
(ii) if Y = CH2, n = 5, m = 7 and R2 = CH3, then R1 is not CF3, CH2Cl, NHOH, C(O)NH2, CN2 or C(O)OEt;
(iii) if Y = CH2Cl, n = 4, m = 7 and R2 = CH3, then R1 is not NH2;
(iv) if Y = CH(OH), n = 4, m = 7 and R2 = CH3, then R1 is not NH2;
(v) if Y = C(O), n = 4, m = 7 and R2 = CH3 , then R1 is not NH2 or CH3CH2O; and
(vi) if Y = CH2, n = 4-9, m = 4-7 and R2 = CH3, then R1 is not NH2 or OH.
ACTIVITY - Neuromodulatory; sleep disorders; mood disorders; analgesia.
MECHANISM OF ACTION - Oleamide agonist; **fatty acid amide hydrolase** inhibitor.
USE - Analogs for oleamide, an endogenous fatty acid primary amide that possesses sleep-inducing properties in animals known to affect serotonergic systems and blocking gap junction communication in a structurally specific manner. Used to inhibit gap junction-mediated chemical and electrical transmission in glial cells (claimed). Used to help distinguish among mechanisms for gap junction inhibition by oleamide and as new neuromodulatory agents applicable to sleep or mood disorders, analgesia, and disorders associated with higher neuronal function. Act as fluidity transmitters.
ADVANTAGE - Storage and release may be controlled in a manner analogous to that of short peptide hormones and messengers terminating in a primary amide.
TECH
ORGANIC CHEMISTRY - Preparation:
(i) tertiary-butyl diphenylsilyl (TBDSP) chloride, triethylamine, 4-N,N'-dimethylaminopyridine (yield 92%);
(ii) triphenylphosphine (yield 96%);
(iii) potassium hexadimethylsilazide; pentadecanal (yield 55%);
(iv) tertiary butyl ammonium fluoride (yield 68%);
(v) pyridinium dichromate, dimethylformamide (yield 76%);
(vi) (COCl)2; ammonium hydroxide (yield 67%).

L30 ANSWER 7 OF 14 WPIX COPYRIGHT 2006 THE THOMSON CORP on STN
DUPLICATE 5
ACCESSION NUMBER: 1997-065456 [06] WPIX

CROSS REFERENCE: 1998-286935
DOC. NO. CPI: C1997-021584 [06]
TITLE: Purified cis-9,10-octadeceno:amidase - useful for hydrolysing sleep-inducing fatty acid primary amide(s), and identifying inhibitors
DERWENT CLASS: B04; B05; D16
INVENTOR: **CRAVATT B F; GILULA N B; LERNER R A**
PATENT ASSIGNEE: (SCRI-C) SCRIPPS RES INST
COUNTRY COUNT: 70

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 9641869	A1	19961227	(199706)*	EN	101[8]	C12N009-78
AU 9661144	A	19970109	(199717)	EN		C12N009-78
EP 833899	A1	19980408	(199818)	EN	[0]	C12N009-78
JP 11507554	W	19990706	(199937)	JA	78	C12N015-00
AU 725984	B	20001026	(200059)	EN		C12N009-78
EP 833899	B1	20050119	(200506)	EN		
DE 69634203	E	20050224	(200516)	DE		
DE 69634203	T2	20060105	(200607)	DE		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9641869 A1		WO 1996-US10435	19960612
AU 9661144 A		AU 1996-61144	19960612
AU 725984 B		AU 1996-61144	19960612
DE 69634203 E		DE 1996-69634203	19960612
EP 833899 A1		EP 1996-918507	19960612
EP 833899 B1		EP 1996-918507	19960612
DE 69634203 E		EP 1996-918507	19960612
EP 833899 A1		WO 1996-US10435	19960612
JP 11507554 W		WO 1996-US10435	19960612
EP 833899 B1		WO 1996-US10435	19960612
DE 69634203 E		WO 1996-US10435	19960612
JP 11507554 W		JP 1997-503386	19960612
DE 69634203 T2		DE 1996-69634203	19960612
DE 69634203 T2		EP 1996-918507	19960612
DE 69634203 T2		WO 1996-US10435	19960612

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 725984 B	Previous Publ	AU 9661144 A
DE 69634203 E	Based on	EP 833899 A
AU 9661144 A	Based on	WO 9641869 A
EP 833899 A1	Based on	WO 9641869 A
JP 11507554 W	Based on	WO 9641869 A
AU 725984 B	Based on	WO 9641869 A
EP 833899 B1	Based on	WO 9641869 A
DE 69634203 E	Based on	WO 9641869 A
DE 69634203 T2	Based on	EP 833899 A
DE 69634203 T2	Based on	WO 9641869 A

PRIORITY APPLN. INFO: US 1995-489535 19950612

INT. PATENT CLASSIF.:
MAIN: C12N015-00; C12N009-78
SECONDARY: A61K031-12; A61K038-46; C12N009-14; C12N009-80; C12N009-99; C12P007-64
IPC RECLASSIF.: A61K0038-43 [,C]; A61K0038-46 [,A]; C12N0009-14 [,A]; C12N0009-78 [,A]; C12N0009-80 [,A]; C12P0007-64 [,A]

BASIC ABSTRACT:
WO 1996041869 A1 UPAB: 20050826 Novel purified form of cis-9,10-octadecenoamidase (COase) is obtd. by a chromatographic methodology selected from affinity, electric, gel filtration, ion exchange and partition chromatography, and is characterised by enzymic activity for catalysing the conversion of cis-9,10-octadecenoamide (CO) to oleic acid and by inclusion of an amino acid sequence selected from (a)-(aa), (a)GGSSGGEGALIGSGGSPLGLGTDIGGSIRFP, (b) SPGGSSGGEGALIGS, (c) ALIGSGGSPLGLGTD, (d) GLGTDIGGSIRFPSA, (e) RFPSAFCGICGLKPT, (f) GLKPTGNRLSKSGLK, (g) KSGLKGCVYGQTAVQ, (h) QTAVQLSLGPMARDV, (i) MARDVESLALCLKAL, (j) CLKALLCEHLFTLDP, (k) FTLDPTVPPFPREE, (l) PFREEVYRSSRPLRV, (m) RPLRVGYYETDNYTM, (n) DNYTMPSPAMRRALI, (o) RRALIETKQRLEAAG, (p) LEAAGHTLIPFLPNN, (q) FLPNNIPYALEVLSA, (r) EVLSAGGLFSDGGRS, (s) DGGRSFLQNKKGDFV, (t) KGDFVDPCLGDLILI, (u) DLILILRLPSWFKRL, (v) WFKRLLSLLLKPLFP, (w) KPLFPRLAAFLNSMR, (x) LNSMRPRSAEKLWKL, (y) KLWKLQHEIEMYRQS, (z) MYRQSVIAQWKAMNL, (aa) KAMNLDVLLTPMLGP, and (ab) PMLGPALDLNTPGR.
USE - The COase can be used to catalyse the hydrolysis of fatty acid primary amides, which have sleep-inducing activity. The COase can also be used to identify inhibitors of the COase activity. MANUAL CODE: CPI: B04-C01; B04-L05; B12-K04A; D05-C03C; D05-H12A; D05-H17A3

Member(0003)
ABEQ EP 833899 A1 UPAB 20050826
Novel purified form of cis-9,10-octadecenoamidase (COase) is obtd. by a chromatographic methodology selected from affinity, electric, gel filtration, ion exchange and partition chromatography, and is characterised by enzymic activity for catalysing the conversion of cis-9,10-octadecenoamide (CO) to oleic acid and by inclusion of an amino acid sequence selected from (a)-(aa), (a)GGSSGGEGALIGSGGSPLGLGTDIGGSIRFP, (b) SPGGSSGGEGALIGS, (c) ALIGSGGSPLGLGTD, (d) GLGTDIGGSIRFPSA, (e) RFPSAFCGICGLKPT, (f) GLKPTGNRLSKSGLK, (g) KSGLKGCVYGQTAVQ, (h) QTAVQLSLGPMARDV, (i) MARDVESLALCLKAL, (j) CLKALLCEHLFTLDP, (k) FTLDPTVPPFPREE, (l) PFREEVYRSSRPLRV, (m) RPLRVGYYETDNYTM, (n) DNYTMPSPAMRRALI, (o) RRALIETKQRLEAAG, (p) LEAAGHTLIPFLPNN, (q) FLPNNIPYALEVLSA, (r) EVLSAGGLFSDGGRS, (s) DGGRSFLQNKKGDFV, (t) KGDFVDPCLGDLILI, (u) DLILILRLPSWFKRL, (v) WFKRLLSLLLKPLFP, (w) KPLFPRLAAFLNSMR, (x) LNSMRPRSAEKLWKL, (y) KLWKLQHEIEMYRQS, (z) MYRQSVIAQWKAMNL, (aa) KAMNLDVLLTPMLGP, and (ab) PMLGPALDLNTPGR.
USE - The COase can be used to catalyse the hydrolysis of fatty acid primary amides, which have sleep-inducing activity. The COase can also be used to identify inhibitors of the COase activity.

Member(0004)
ABEQ JP 11507554 W UPAB 20050826
Novel purified form of cis-9,10-octadecenoamidase (COase) is obtd. by a chromatographic methodology selected from affinity, electric, gel filtration, ion exchange and partition chromatography, and is characterised by enzymic activity for catalysing the conversion of cis-9,10-octadecenoamide (CO) to oleic acid and by inclusion of an amino acid sequence selected from (a)-(aa), (a)GGSSGGEGALIGSGGSPLGLGTDIGGSIRFP, (b) SPGGSSGGEGALIGS, (c) ALIGSGGSPLGLGTD, (d) GLGTDIGGSIRFPSA, (e) RFPSAFCGICGLKPT, (f) GLKPTGNRLSKSGLK, (g) KSGLKGCVYGQTAVQ, (h) QTAVQLSLGPMARDV, (i) MARDVESLALCLKAL, (j) CLKALLCEHLFTLDP, (k)

FTLDPTVPPFPREE, (l) PFREEVYRSSRPLRV, (m) RPLRVGYYETDNYTM, (n) DNYTMPSPAMRRALI, (o) RRALIETKQRLEAAG, (p) LEAAGHTLIPFLPNN, (q) FLPNNIPYALEVLSA, (r) EVLSAGGLFSDGGRS, (s) DGGRSFLQNKKGDFV, (t) KGDFVDPCLGDLILI, (u) DLILILRLPSWFKRL, (v) WFKRLLSLLLKPLFP, (w) KPLFPRLAAFLNSMR, (x) LNSMRPRSAEKLWKL, (y) KLWKLQHEIEMYRQS, (z) MYRQSVIAQWKAMNL, (aa) KAMNLDVLLTPMLGP, and (ab) PMLGPALDLNTPGR.

USE - The COase can be used to catalyse the hydrolysis of fatty acid primary amides, which have sleep-inducing activity. The COase can also be used to identify inhibitors of the COase activity.

L30 ANSWER 8 OF 14 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:186194 BIOSIS Full-text
DOCUMENT NUMBER: PREV200400190623
TITLE: Assay for inhibitors of **fatty-acid amide hydrolase.**
AUTHOR(S): **Gilula, Norton B.** [Inventor, Reprint Author]; **Cravatt, Benjamin F.** [Inventor]; **Lerner, Richrd A.** [Inventor]
CORPORATE SOURCE: La Jolla, CA, USA
ASSIGNEE: The Scripps Research Institute
PATENT INFORMATION: US 6699682 20040302
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Mar 2 2004) Vol. 1280, No. 1. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2004
Last Updated on STN: 7 Apr 2004

ABSTRACT:The soporific activity of cis-9,10-octadecenoamide and other soporific fatty acid primary amides is neutralized by hydrolysis in the presence of ***fatty*** **-acid amide hydrolase (FAAH**). Hydrolysis of cis-9,10-octadecenoamide by **FAAH** leads to the formation of oleic acid, a compound without soporific activity. **FAAH** has be isolated and the gene encoding **FAAH** has been cloned, sequenced, and used to express recombinant **FAAH**. Inhibitors of ***FAAH*** are disclosed to block the hydrolase activity.

NAT. PATENT. CLASSIF.:435018000
CONCEPT CODE: Genetics - General 03502
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Pathology - Therapy 12512
Pharmacology - General 22002
INDEX TERMS: Major Concepts
Methods and Techniques; Molecular Genetics (Biochemistry and Molecular Biophysics); Pharmacology
INDEX TERMS: Chemicals & Biochemicals
cis-9,10-octadecenoamide: soporific activity; **fatty-acid amide hydrolase; fatty-acid amide hydrolase** inhibitors: enzyme inhibitor-drug; soporific fatty acid primary amides
INDEX TERMS: Methods & Equipment
fatty-acid amide hydrolase inhibitor assay: laboratory techniques
REGISTRY NUMBER: 301-02-0 (cis-9,10-octadecenoamide)
153301-19-0 (**fatty-acid amide**

hydrolase)
GENE NAME: **FAAH** gene [**fatty-acid amide hydrolase** gene]: nucleotide sequence

L30 ANSWER 9 OF 14 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1997:142732 BIOSIS Full-text
DOCUMENT NUMBER: PREV199799441935
TITLE: Electron cryo-crystallography reveals that oleamide, a sleep inducing compound, causes structural changes of a gap junction channel.
AUTHOR(S): Unger, V. M.; Entrikin, D. W.; Guan, X.; **Cravatt, B.**; Kumar, N. M.; **Lerner, R. A.**; **Gilula, N. B.**; Yeager, M.
CORPORATE SOURCE: Scripps Res. Inst., La Jolla, CA 92037, USA
SOURCE: Biophysical Journal, (1997) Vol. 72, No. 2 PART 2, pp. A291.
Meeting Info.: 41st Annual Meeting of the Biophysical Society. New Orleans, Louisiana, USA. March 2-6, 1997.
CODEN: BIOJAU. ISSN: 0006-3495.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Apr 1997
Last Updated on STN: 2 May 1997
CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Lipids 10066
Biophysics - Molecular properties and macromolecules 10506
Biophysics - Membrane phenomena 10508
INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Membranes (Cell Biology)
INDEX TERMS: Chemicals & Biochemicals
OLEAMIDE
INDEX TERMS: Miscellaneous Descriptors
ALPHA-CONNEXIN; ANALYTICAL METHOD; BHK CELL LINE; BIOCHEMISTRY AND BIOPHYSICS; CRYSTALLOGRAPHY; GAP JUNCTION CHANNEL; LIPID; MEMBRANES; OLEAMIDE; SLEEP-INDUCING COMPOUND; STRUCTURAL CHANGES
ORGANISM: Classifier
Cricetidae 86310
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Cricetidae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
REGISTRY NUMBER: 301-02-0 (OLEAMIDE)

L30 ANSWER 10 OF 14 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1997:95938 BIOSIS Full-text
DOCUMENT NUMBER: PREV199799395141

TITLE: The sleep induction lipid oleamide blocks gap junction communication but not calcium wave transmission in glial cells.
AUTHOR(S): Guan, X. J. [Reprint author]; **Cravatt, B. F.**; Ehring, G. R.; Hall, J. E.; Boger, D. L.; **Lerner, R. A.**; **Gilula, N. B.** [Reprint author]
CORPORATE SOURCE: Dep. Cell Biol., The Scripps Res. Inst., La Jolla, CA 92307, USA
SOURCE: Molecular Biology of the Cell, (1996) Vol. 7, No. SUPPL., pp. 280A.
Meeting Info.: Annual Meeting of the 6th International Congress on Cell Biology and the 36th American Society for Cell Biology. San Francisco, California, USA. December 7-11, 1996.
CODEN: MBCEEV. ISSN: 1059-1524.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Mar 1997
Last Updated on STN: 2 Apr 1997
CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - General 10060
Biophysics - General 10502
Nervous system - General and methods 20501
INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Nervous System (Neural Coordination)
INDEX TERMS: Chemicals & Biochemicals
OLEAMIDE; CALCIUM
INDEX TERMS: Miscellaneous Descriptors
CALCIUM WAVE TRANSMISSION; GAP JUNCTION; GAP JUNCTION COMMUNICATION; GLIAL CELL; MEMBRANES; NERVOUS SYSTEM; OLEAMIDE; SLEEP INDUCING LIPID
ORGANISM: Classifier
Felidae 85770
Super Taxa
Carnivora; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
cat
Taxa Notes
Animals, Carnivores, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
REGISTRY NUMBER: 301-02-0 (OLEAMIDE)
7440-70-2 (CALCIUM)

L30 ANSWER 11 OF 14 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:204353 BIOSIS Full-text
DOCUMENT NUMBER: PREV199698760482
TITLE: An endogenous sleep-inducing lipid activates intercellular calcium waves in glial cells.
AUTHOR(S): Charles, A. C. [Reprint author]; **Cravatt, B. F.**; Boger, D. L.; **Gilula, N. B.** [Reprint author]; **Lerner, R. A.**
CORPORATE SOURCE: Dep. Neurol., UCLA, Los Angeles, CA 90095, USA
SOURCE: Journal of Neurochemistry, (1996) Vol. 66, No. SUPPL. 1, pp. S75.

Meeting Info.: 27th Annual Meeting of the American Society for Neurochemistry. Philadelphia, Pennsylvania, USA. March 2-6, 1996.
CODEN: JONRA9. ISSN: 0022-3042.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 2 May 1996
Last Updated on STN: 2 May 1996

CONCEPT CODE: General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Behavioral biology - Animal behavior 07003
Biochemistry methods - Proteins, peptides and amino acids 10054
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Molecular properties and macromolecules 10506
Biophysics - Membrane phenomena 10508
Metabolism - Minerals 13010
Metabolism - Proteins, peptides and amino acids 13012
Endocrine - Neuroendocrinology 17020
Nervous system - General and methods 20501
Nervous system - Physiology and biochemistry 20504
Psychiatry - Psychophysiology 21003

INDEX TERMS: Major Concepts
Behavior; Biochemistry and Molecular Biophysics; Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Metabolism; Nervous System (Neural Coordination)

INDEX TERMS: Chemicals & Biochemicals
CALCIUM

INDEX TERMS: Miscellaneous Descriptors
CELLULAR SIGNAL; CIS-9,10-OCTADECENOAMIDE; MEETING ABSTRACT; RAT BRAIN

ORGANISM: Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Muridae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

REGISTRY NUMBER: 7440-70-2 (CALCIUM)

L30 ANSWER 12 OF 14 CONFSCI COPYRIGHT 2006 CSA on STN

ACCESSION NUMBER: 96:27175 CONFSCI

DOCUMENT NUMBER: 96-039048

TITLE: Endogenous sleep-inducing lipid activates intercellular calcium waves in glial cells

AUTHOR: Charles, A.C.; **Cravatt, B.F.**; Boger, D.L.; **Gilula, N.B.**; **Lerner, R.A.**

CORPORATE SOURCE: Dep. Neurology, UCLA, Los Angeles, CA 90095, USA

SOURCE: American Society for Neurochemistry, 301 University Boulevard, MRB 2.143, Galveston, TX 77555-1069, Abstracts and full papers available..
Meeting Info.: 961 0209: 27th Annual Meeting of the

American Society for Neurochemistry (9610209). Philadelphia , PA (USA). 2-7 Mar 1996. American Society for Neurochemistry.
DOCUMENT TYPE: Conference
FILE SEGMENT: DCCP
LANGUAGE: English
CLASSIFICATION: 2500 CHEMISTRY AND CHEMICAL ENGINEERING

L30 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:569722 CAPLUS Full-text
DOCUMENT NUMBER: 135:164083
TITLE: Characterization of mammalian fatty acid amide hydrolases and their role in the metabolism of soporific fatty acid primary amides
INVENTOR(S): **Gilula, Norton B.; Cravatt, Benjamin F.; Lerner, Richard A.**
PATENT ASSIGNEE(S): The Scripps Research Institute, USA
SOURCE: U.S., 72 pp., Cont.-in-part of U.S. Ser. No. 489,535, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6271015	B1	20010807	US 1996-743168	19961104
CA 2224426	A1	19961227	CA 1996-2224426	19960612
CA 2270293	A1	19980514	CA 1997-2270293	19971104
WO 9820119	A1	19980514	WO 1997-US20385	19971104
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9854321	A	19980529	AU 1998-54321	19971104
AU 739962	B2	20011025		
EP 970195	A1	20000112	EP 1997-948210	19971104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2001503630	T	20010321	JP 1998-521812	19971104
US 2002187542	A1	20021212	US 2001-894790	20010628
US 6699682	B2	20040302		
AU 781382	B2	20050519	AU 2002-13606	20020125
US 2004265958	A1	20041230	US 2004-788992	20040226

PRIORITY APPLN. INFO.:

US 1995-489535	B2	19950612
WO 1996-US10435	A2	19960612
US 1996-743168	A	19961104
AU 1998-54321	A3	19971104
WO 1997-US20385	W	19971104
US 2001-894790	A3	20010628

ED Entered STN: 07 Aug 2001

AB The soporific activity of cis-9,10-octadecenoamide and other soporific fatty acid primary amides is neutralized by hydrolysis in the presence of fatty acid amide hydrolase (FAAH, also known as cis-9,10-octadecenoamidase or oleamide amidase). Hydrolysis of cis-9,10-octadecenoamide by FAAH leads to the formation of oleic acid, a compound without soporific activity. FAAH was isolated and the gene encoding rat, mouse, and human FAAH was cloned, sequenced, and used to express recombinant FAAH. Inhibitors of FAAH are disclosed to block the hydrolase activity.

IC ICM C12N009-80

INCL 435228000

CC 7-2 (Enzymes)
Section cross-reference(s): 1, 3

ST **fatty acid amide hydrolase**
soporific metab mammal; sequence **fatty acid amide hydrolase** cDNA mammal

IT 153301-19-0P, **Fatty acid amide hydrolase**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation); PROC (Process)
(characterization of mammalian fatty acid amide hydrolases and their role in the metabolism of soporific fatty acid primary amides)

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:324876 CAPLUS Full-text
DOCUMENT NUMBER: 129:25077
TITLE: Cloning of mammalian fatty-acid amide hydrolases active on octadecenoamide sleep inducers
INVENTOR(S): **Gilula, Norton B.; Cravatt, Benjamin F.; Lerner, Richard A.**
PATENT ASSIGNEE(S): Scripps Research Institute, USA
SOURCE: PCT Int. Appl., 151 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9820119	A1	19980514	WO 1997-US20385	19971104
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6271015	B1	20010807	US 1996-743168	19961104
CA 2270293	A1	19980514	CA 1997-2270293	19971104
AU 9854321	A	19980529	AU 1998-54321	19971104
AU 739962	B2	20011025		
EP 970195	A1	20000112	EP 1997-948210	19971104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2001503630	T	20010321	JP 1998-521812	19971104

AU 781382 B2 20050519 AU 2002-13606 20020125
PRIORITY APPLN. INFO.: US 1996-743168 A 19961104
US 1995-489535 B2 19950612
AU 1998-54321 A3 19971104
WO 1997-US20385 W 19971104

OTHER SOURCE(S): MARPAT 129:25077
ED Entered STN: 01 Jun 1998
AB The soporific activity of cis-9,10-octadecenoamide and other soporific fatty-acid primary amides is neutralized by hydrolysis in the presence of fatty-acid amide hydrolase (FAAH). Hydrolysis of cis-9,10-octadecenoamide by FAAH leads to the formation of oleic acid, a compound without soporific activity. FAAH was isolated and the gene encoding FAAH was cloned, sequenced, and used to express recombinant FAAH. FAAH was purified from rat liver (20-30-fold enriched, 10-15% yield) by 4 steps of column chromatog. CDNAs were isolated from rat, mouse, and human, and their nucleotide and deduced amino acid sequence provided. Inhibitors of FAAH are disclosed to block the hydrolase activity.
IC ICM C12N009-78
ICS C12N009-80; C12N009-14; C12P007-64; A16K038-46
CC 7-2 (Enzymes)
Section cross-reference(s): 1, 3
IT 183869-93-4P 186814-44-8P 189122-02-9P, **Fatty acid amide hydrolase** (human) 208065-35-4P, Amidase (mouse)
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; cloning of mammalian fatty-acid amide hydrolases active on octadecenoamide sleep inducers)
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

FILE 'HOME' ENTERED AT 11:48:40 ON 27 DEC 2006

SEARCH HISTORY

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=> d his nofile

     (FILE 'HOME' ENTERED AT 11:28:01 ON 27 DEC 2006)

     FILE 'STNGUIDE' ENTERED AT 11:28:11 ON 27 DEC 2006

     FILE 'MEDLINE, PASCAL, CABA, WPIX, BIOTECHNO, BIOSIS, LIFESCI, CONFSCI,
     CAPLUS, DISSABS, EMBASE' ENTERED AT 11:30:54 ON 27 DEC 2006
L1          619 SEA ABB=ON  GILULA N/AU OR GILULA NB/AU OR GILULA N B/AU OR
                GILULA NORTON?/AU
L2          673 SEA ABB=ON  CRAVATT B/AU OR CRAVATT BF/AU OR CRAVATT B F/AU OR
                CRAVATT BENJAMIN?/AU
L3         2601 SEA ABB=ON  LERNER R/AU OR LERNER RA/AU OR LERNER R A/AU OR
                LERNER RICH?/AU
L4            3 SEA ABB=ON  FATTY ACID AMINE HYDROLASE
L5         1164 SEA ABB=ON  FAAH
L6           35 SEA ABB=ON  L1 AND L2 AND L3
L7          250 SEA ABB=ON  (L1 OR L2 OR L3) AND (L4 OR L5)
L8            0 SEA ABB=ON  (L1 OR L2 OR L3) AND L4
L9            2 SEA ABB=ON  L6 AND L5
                D KWIC 1-2
L10        1837 SEA ABB=ON  FATTY-ACID AMIDE HYDROLASE
L11         331 SEA ABB=ON  (L1 OR L2 OR L3) AND L10
L12           2 SEA ABB=ON  (L5 OR L10) AND (PY<1995 OR AY<1995 OR PRY<1995)
L13      951241 SEA ABB=ON  HYDROLY?
L14          77 SEA ABB=ON  L11 AND L13
L15          25 SEA ABB=ON  ((L1 AND (L2 OR L3)) OR (L2 AND L3)) AND (L5 OR
                L10)
L16         589 SEA ABB=ON  MAMMAL? AND (L5 OR L10)

     FILE 'MEDLINE, PASCAL, CABA, WPIX, BIOTECHNO, BIOSIS, LIFESCI, CONFSCI,
     CAPLUS, DISSABS, EMBASE' ENTERED AT 11:41:09 ON 27 DEC 2006
                D QUE L12
L17           2 DUP REM L12 (0 DUPLICATES REMOVED)
                      ANSWER '1' FROM FILE MEDLINE
                      ANSWER '2' FROM FILE WPIX
                D IALL 1
                D IALL ABEQ TECH 2
L18        8619 SEA ABB=ON  OCTACECENOAMIDE OR ANANDAMIDE OR (MYRISTIC OR
                PALMITIC OR STEARIC)(W) AMIDE
L19      171633 SEA ABB=ON  VECTOR# AND RECOMB?
L20         309 SEA ABB=ON  (L5 OR L10) AND L18(10A) L13
L21         224 SEA ABB=ON  (L5 OR L10) AND L18(3A) L13
L22          15 SEA ABB=ON  L19 AND (L5 OR L10)
L23     2143366 SEA ABB=ON  CHROMATOG?
L24           7 SEA ABB=ON  L23 AND L21
L25          20 SEA ABB=ON  L23 AND L20
                D QUE L22
                D QUE L25
L26          34 SEA ABB=ON  (L22 OR L25) NOT (L6 OR L15)
L27          20 DUP REM L26 (14 DUPLICATES REMOVED)
                      ANSWERS '1-10' FROM FILE MEDLINE
                      ANSWERS '11-14' FROM FILE WPIX
                      ANSWER '15' FROM FILE BIOTECHNO
                      ANSWERS '16-17' FROM FILE BIOSIS
                      ANSWER '18' FROM FILE CAPLUS
                      ANSWERS '19-20' FROM FILE DISSABS
                D IALL 1-10
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                D IALL ABEQ TECH 11-14
                D IALL 15-17
                D IBIB ED ABS HITIND 18
                D IALL 19-20

     FILE 'MEDLINE, PASCAL, CABA, WPIX, BIOTECHNO, BIOSIS, LIFESCI, CONFSCI,
     CAPLUS, DISSABS, EMBASE' ENTERED AT 11:46:58 ON 27 DEC 2006
                D QUE L12
                D QUE L6
                D QUE L15
L28          41 SEA ABB=ON  (L6 OR L15)
L29          40 SEA ABB=ON  L28 NOT (L22 OR L25)
L30          14 DUP REM L29 (26 DUPLICATES REMOVED)
                     ANSWERS '1-5' FROM FILE MEDLINE
                     ANSWERS '6-7' FROM FILE WPIX
                     ANSWERS '8-11' FROM FILE BIOSIS
                     ANSWER '12' FROM FILE CONFSCI
                     ANSWERS '13-14' FROM FILE CAPLUS
                D IALL 1-5
                D IALL ABEQ TECH 6-7
                D IALL 8-12
                D IBIB ED ABS HITIND 13-14

     FILE 'HOME' ENTERED AT 11:48:40 ON 27 DEC 2006

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